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The impact of childhood sickness on adult socioeconomic outcomes: Evidence from late 19th century America

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Abstract

We use family fixed-effects models to estimate the impact of childhood health on adult literacy, labor force outcomes, and marital status among pairs of white brothers observed as children in the 1880 U.S. Census and then as adults in the 1900–1930 Censuses. Given our focus on the 19th century, we observed a wider array of infectious, chronic, and traumatic health problems than is observed using data that are more recent; our results thus provide some insights into circumstances in modern developing countries where similar health problems are more frequently observed. Compared to their healthy siblings, sick brothers were less likely to be located (and thus more likely to be dead) 20–50 years after their 1880 enumeration. Sick brothers were also less likely to be literate, to have ever been married, and to have reported an occupation. However, among those with occupations, sick and healthy brothers tended to do similar kinds of work. We discuss the implications of our results for research on the impact of childhood health on socioeconomic outcomes in developed and developing countries.

Keywords

Health inequalities; Childhood health; Socioeconomic status; Developing countries; Historical demography; U.S.A

Introduction

Research using data from developed, high-income countries shows that childhood health has long-term consequences for educational attainment and adult socioeconomic circumstances (e.g., Case, Fertig, & Paxson, 2005; Currie, 2009; Haas, 2006; Haas, Glymour, & Berkman, 2011; Jackson, 2010; Palloni, 2006; Palloni, Milesi, White, & Turner, 2009). For example, after noting that "disadvantaged social background is associated with poor childhood health" in the contemporary United States, Haas (2006: 339) concluded that "poor health in childhood has significant, direct, and large adverse effects" on educational attainment, occupational standing, earnings, and wealth accumulation.

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Appendix A. Supplementary data: Supplementary data related to this article can be found online at http://dx.doi.Org/10.1016/j.socscimed.2012.06.001.

With only a few exceptions, evidence for the effects of childhood health on subsequent socioeconomic outcomes has come from studies using late 20th-century or early 21st-century data on individuals in developed countries. As described in more detail below, this evidence is limiting in three important ways. First, it tells us relatively little about the impact of childhood health on adult outcomes in contemporary developing countries, where prevalence rates and disease profiles of childhood illness, patterns of educational enrollment and attainment, welfare state institutions, and labor market processes are markedly different from those in developed countries. Second, the late 20th and early 21st centuries are (fortunately) characterized by relatively low childhood morbidity rates in developed countries, and the health problems that most frequently affect children in those countries tend to be chronic in nature. As a result, even studies that are adequately powered to detect effects of childhood health cannot investigate the impacts of different types of diseases or health problems (e.g., specific infectious diseases or traumatic injuries) on later health or status attainment. Third, because their research has frequently been based on information about unrelated individuals, many recent investigators could not rule out the possibility that apparent effects of childhood health on subsequent socioeconomic outcomes are spurious owing to unobserved (family, genetic, environmental, or other) factors that might impact both childhood health and adult socioeconomic outcomes. We follow research in economics and elsewhere (e.g., Black, Devereux, & Salvanes, 2005; Conley & Bennett, 2000; Currie, 2009; Haas, 2006; Johnson & Schoeni, 2007) that reduces the threat of this methodological problem by modeling data on siblings.

In this article, we utilize data from the late 19th century United States to assess the impact of childhood health on three sets of social and economic outcomes among white men: literacy, labor force outcomes, and marital status. Our results are useful for understanding the role of childhood health in stratifying social and economic outcomes in an important era in American history. As we discuss below, our results also provide insights for scholars who seek to understand better the long-term impacts of childhood health problems in contemporary developing countries. Beyond this, our results are useful for two other reasons. First, our estimates are unlikely to be seriously biased owing to unobserved confounders because we focus on brothers raised in the same household. Second, like children in contemporary developing countries, the brothers in our 19th century American sample suffered in large numbers from a wide array of infectious, chronic, and traumatic health problems.

We begin by reviewing relevant theoretical and empirical background on the impact of childhood health on socioeconomic outcomes. We then detail our critique of the empirical literature on the grounds outlined above, and explain how our empirical project improves upon the extant literature. Finally, after describing our data and empirical strategy, we present our results and discuss their potential relevance for research on the contemporary impact of childhood health on socioeconomic outcomes in the developed and developing world.

Theoretical background

Research interest in the socioeconomic consequences of childhood health grew out of a broader effort to understand the sources of well-documented social and economic gradients in adult morbidity and mortality. The predominant explanation for such gradients has been that socioeconomic standing causally influences health (via mechanisms that are not entirely understood). A competing (or perhaps supplementary) theoretical explanation is that health problems shunt individuals into lower socioeconomic positions via one of two hypothesized pathways. First, the “socioeconomic drift” pathway supposes that the onset of serious health problems in adulthood harms people's socioeconomic circumstances by draining their financial resources (e.g., spending previously accumulated assets to pay for treatment or to make up for lost wages) and limiting their capacity to succeed in the labor market. Second, the "social stunting" pathway suggests that health problems early in life limit people's ability to acquire the skills and human capital that are necessary to achieve advantaged socioeconomic positions. Palloni et al. (2009) and Haas et al. (2011) provide more thorough discussions of these several hypotheses. Thus health selection can operate in two ways, either by inducing downward social mobility (i.e., drift) or by preventing the initial movement upward (i.e., stunting). The extant health inequality literature provides evidence that both processes occur in various contexts over the life course.

Research in the "social stunting" tradition has focused on the impact of early life health conditions on educational and labor market outcomes. Measured in terms of low birth weight status, self-assessed overall health, or counts of chronic conditions, childhood health has been linked to academic achievement and to the quantity of completed schooling (e.g., Boardman, Powers, Padilla, & Hummer, 2002; Case et al., 2005; Conley & Bennett, 2000; Haas & Fosse, 2008; Jackson, 2010; Palloni, 2006; Wadsworth, 1991). In addition to educational attainment, childhood health also influences adult earnings (e.g., Black et al., 2005; Currie, 2009; Haas, 2006; Haas et al., 2011; Johnson & Schoeni, 2007) and occupational attainment and social class (e.g., Case et al., 2005; Jackson, 2010; Palloni, 2006; Palloni et al., 2009), perhaps largely via its influence on cognitive and non-cognitive skills and educational attainment.

Our work builds on the "social stunting" perspective by investigating such processes in a very different socio-historical and epidemiologic context than has been previously examined. In late 19th century America, both the discrete markers of socioeconomic success and the pathways to getting there were different than they are now. In 1880, a growing number of men were finding wage-paying manufacturing or other blue-collar jobs in cities; at the same time, about half worked on farms. Formal education beyond primary schooling was quite rare. Outside of basic literacy, "getting ahead" did not generally require much formal schooling.

In this context, how might "social stunting" have played out? Childhood illnesses and disabilities, especially if they limited boys for long periods, may have hindered the acquisition of basic forms of late 19th century human capital. As in contemporary contexts, that human capital was partly in the form of knowledge (i.e., basic literacy and numeracy). However, in that era human capital in the form of farming or trade skills was also at least as important. We hypothesize that childhood illnesses limited boys' literacy and/or their acquisition of knowledge and experience about farming or specific trades. That, in turn, affected whether those boys were able to support themselves as adults through work; it also affected the kind of work that they were able to do. What is more, given the division of household labor of that era, we expect that men with limited means to support themselves through work would have been less likely to marry. Because infectious and chronic illness and traumatic injuries all would have different implications for boys' ability to acquire these forms of human capital, we expect to see that not all health problems mattered equally for men's socioeconomic outcomes in the 19th century. In particular, we hypothesize that chronic diseases and traumatic injuries—both of which imply longer-term disruptions in human capital acquisition—had the greatest impacts. To summarize, the following hypotheses guide our empirical work:

> Hypothesis 1: Childhood illnesses limited boys' literacy and acquisition of human capital.
>
> Hypothesis 2: Childhood illnesses negatively affected the type of work men were able to do in adulthood, reducing their labor force participation.

Hypothesis 3: Childhood illnesses negatively affected the type of work men were able to do in adulthood, reducing their occupational standing.

Hypothesis 4: Childhood illnesses reduced men's ability to support themselves through work and thus they were less likely to marry.

Hypothesis 5: Childhood chronic diseases and traumatic injuries had a greater impact than infectious diseases on later human capital accumulation.

Empirical background

The existing empirical literature on the impact of childhood health on adult socioeconomic outcomes is limited in several important respects. Much of what is known about the long-term socioeconomic consequences of poor childhood health is derived from cohort studies in developed countries in the late 20th and early 21st century. Research on these effects in the context of contemporary developing countries has been largely constrained to investigating the impact of childhood malnutrition (e.g., Alderman, Hoddinott, & Kinsey, 2006; Chen & Zhou, 2007; Field, Robles, & Torero, 2009; Hoddinott, Maluccio, Behrman, Flores, & Martorell, 2008; Maluccio et al., 2009). For example, a systematic review of evidence from intervention and prospective cohort studies in Brazil, India, the Philippines, South Africa, and Guatemala found that childhood under-nutrition was associated with reduced cognitive performance, delayed school entry, grade repetition, dropout, reduced completed schooling, and lower subsequent wages, household income, and assets (Victora et al., 2008). Beyond this work on malnutrition, however, we have very little evidence about the impact of childhood health on subsequent social and economic well-being in contemporary developing countries.

In addition, previous investigators' nearly exclusive focus on contemporary developed countries is limiting because of the prevalence rates and disease profiles that characterize modern societies. Put simply, relatively few children are sick in developed countries, and those who are sick generally suffer from chronic diseases. In order to assess the impact of childhood health on subsequent outcomes in developed countries, researchers using contemporary longitudinal data must have access to relatively large samples (because of low prevalence rates) and they must focus on a narrow range of childhood health indicators (typically low birth weight status, self-assessed overall health, or counts of chronic diseases). For instance, using longitudinal data on developed countries from the past half-century, researchers cannot assess the long-term impacts of infectious childhood diseases or traumatic injuries. At the same time—and even though infectious conditions represent a larger burden of disease in developing countries—there is very little high quality population-based data in developing countries that are suitable for understanding the impact of childhood infectious diseases. Thus, for different reasons, contemporary data in both developing and developed countries are ill suited to investigate the long-term socioeconomic impacts of infectious disease. They are especially ill suited for studying the relative effects of different kinds of health problems.

The analysis of historical data from *developed* countries is a common method for shedding light on processes that currently affect *developing* countries. For example, malaria exposure and mortality rates in the early 20th century in parts of the southern U.S. were comparable to those currently observed in many developing countries. Previous research has successfully used data from the early 20th century United States to investigate the long-term impacts of fetal and post-natal malaria exposure on educational outcomes (Barreca, 2010). Similarly, Almond (2006) and Mazumder, Almond, Park, Crimmins, and Finch (2010) used historical data from 1918 Spanish Influenza pandemic to investigate the impact of early life exposures to adult health and socioeconomic attainment.

An important structural difference between developed and developing countries is the presence of opportunities for the remediation of childhood health insults. At the household level, families in developing countries must already cope with scarce resources for investing in the human capital of their children. Devoting additional resources to ameliorate further the effects of health insults may be prohibitive. Such constraints are mirrored at the national level as developing countries often lack well-established welfare states and civil society institutions to provide a social safety net that may otherwise be able to compensate for health insults in ways that poor families cannot. Conversely, in developed countries, the welfare state can and often does fill the investment void among disadvantaged families, acting to moderate the impact of a wide variety of childhood insults and deprivations (Chung & Muntaner, 2006; Navarro & Shi, 2001).

Even though there are obvious differences, in some ways the late 19th century United States resembled contemporary developing countries in a number of important respects. In addition to predating the development of the welfare state, the late 19th century United States was also an environment characterized by rapid industrialization and urbanization and nascent or nonexistent public health systems. Perhaps most importantly, it was also undergoing—albeit at a different rate—similar fundamental demographic and epidemiologic transformations that many developing countries are now experiencing (Omran, 1971). In this way, our empirical work on the impact of childhood health on social and economic outcomes in the United States in the late 19th century can inform our understanding of the impact of childhood health on parallel outcomes in contemporary developing countries.

Of course, there are important limits to such comparisons. Industrialization in modern developing countries is occurring at a different pace and under very different geopolitical, technologic, and economic circumstances than in late 19th century U.S. Unlike in the U.S. in the 19th century, the limited public health systems in modern developing countries are supplemented by the efforts of innumerable non-governmental organizations. Moreover, modern nations are developing during a time when they have access to biomedical information about the etiology of prevalent infectious and chronic diseases, information that was not available a century ago in the U.S. In addition, whereas the demographic transition in the U.S. occurred over more than a century, in some contemporary developing countries it has occurred within the span of a generation. Nonetheless, while it is important to note the structural differences between these two contexts, we argue that at an individual-level many of the processes operate similarly.

An important limitation of previous research based on historical samples is that they often lack a direct observation of childhood health. Many researchers have thus employed proxies of early life infectious disease exposure such as month and year of birth (e.g., Doblhammer & Vaupel, 2001), often in conjunction with exogenous ecological variables or policy changes that are useful for deriving instrumental variable estimates of the impacts of childhood exposures (e.g., Almond, 2006; Barreca, 2010). In addition, traditional estimates based on covariate adjustment may be at least partially spurious owing to unobserved social, economic, environmental, and biological confounders. For example, there is considerable evidence that children's social and economic circumstances have important implications for early life health outcomes (e.g., Haas, 2006; Nikiema, Spencer, & Seguin, 2010; Spencer, 2003; Strully, Rehkopf, & Xuan, 2010) and for subsequent socioeconomic attainments. Thus, unobserved prior causes may bias estimates of the effect of childhood health. Our analysis combines a direct assessment of health in childhood with a within-family estimation strategy to account for unobserved fixed family-level social and biological factors that may generate spurious associations.

In our empirical project, we utilize longitudinal data on sibling pairs observed in the late 19th century United States. Although full siblings are not identical with respect to genetic and socioeconomic backgrounds, they are much more similar than unrelated individuals are and thus the risk of confounding is substantially reduced. By using longitudinal data from the late 19th century United States, we observe sizable numbers of children suffering from maladies that are relatively rare in developed countries but that remain common sources of morbidity and mortality in developing countries (e.g., malaria, measles, tuberculosis, and traumatic injuries). Beyond allowing us to assess the impact of different types of health problems in the U.S., this analytic focus means that our results speak—albeit with some qualifications—to the impact of childhood health in contemporary developing countries. Our work contributes to the theoretical and empirical literature reviewed above by using a sound estimation strategy and by focusing on an era in which disease profiles and prevalence rates were markedly different.

Research design

The 1880 U.S. Census is the only surviving U.S. federal census to ascertain the nature of individuals' illness or disability. Of the roughly 50 million residents enumerated, more than 500,000 reported some health problem that limited their ability to attend to ordinary business or duties; most of the afflicted suffered from an infectious disease (Elman & Myers, 1997). In a project executed jointly with the Church of Jesus Christ of Latter-day Saints, the Minnesota Population Center's Integrated Public Use Microdata Series (IPUMS; Ruggles et al., 2010) created 100%, 10%, and 1% random samples from the 1880 Census files. Because the 100% sample does not include the focal health variable, we only make use of the 10% of 1% samples.

From these files, we randomly selected 2500 households that contained (1) one or more child of the household head who had some illness or disability and (2) one or more child of the household head who had no such illness or disability. Within households, we then randomly selected one child who had an illness or disability and one who did not. We refer to these two children as “siblings” even though we do not know with certainty that they are actually related in this way. They are both children of the same household head, and in all but a handful of cases, they are similar in age and share the same last name as the household head. We suspect that the vast majority of pairs are, in fact, siblings. Note that this procedure was implemented in such a way that the small number of brother pairs who lived together but who were not children of the household head could not have been selected into our sample.

For reasons more fully described in the online Appendix, we were unable to link 1880 to 1900–1930 U.S. census records for women (due to a lack of information about name changes after marriage) or for African Americans (due to high rates of migration and ambiguity about birth dates). After excluding 352 pairs of African American brothers from our initial sample of 2500 brother pairs, our analytic sample includes 2148 pairs of white brothers, one of whom had an illness or disability in 1880 and one of whom did not.

Our procedures for linking 1880 to 1900–1930 census records are described in the online Appendix, and are based on the availability of full identifying information in the public-use 1880 Census files and the resources of the genealogical website Ancestry.com. As described more fully in the Appendix, using conservative procedures we were able to successfully link records for 61% of individual white boys and for 45% of complete white brother pairs (n = 962 pairs); many unlinked boys likely died before 1900. These rates of linkage are as good or better than other efforts to link 19th century American census records (e.g., Ferrie, 1996; Guest, Landale, & McCann, 1989). As shown in online Appendix Table A1, linked boys resemble unlinked boys on observable dimensions. Further details about our linking efforts are available upon request.

Independent variables

Our main independent variable is based on the information collected in Column 15 of the 1880 U.S. Census form. For every individual, enumerators asked the head of the household:

“Is the person [on the day of the enumerator's visit] sick or temporarily disabled, so as to be unable to attend to ordinary business or duties? If so, what is the sickness or disability?”

This variable is known to undercount illnesses and disabilities in two ways. First, because some respondents took “ordinary business or duties” to mean *work-related* business or duties, and because most young children did not have work-related responsibilities, health problems were underreported for young children (Elman & Myers, 1997, 1999). Second, because the question implicitly asks about health problems that *prevent* attention to ordinary business or duties—as opposed to health problems that simply make attention to those things *more difficult*—sickness and disability may have been underreported among people who persevered in their business or duties despite their health problems. Finally, we recognize the inherent measurement problems associated with self-reports of disease and disability.

If childhood sickness were simply underreported across the board, this would not affect our estimates of the effects of childhood sickness on subsequent outcomes. Of potentially greater concern is differential underreporting of childhood sickness across socioeconomic groups. However, we suspect that this form of non-random measurement error leads us to *understate* the effects of childhood health. If we were focusing primarily on *between-family* comparisons—that is, if our identification of the effects of childhood health were based on comparisons of healthy and sick boys *across* families—then differential underreporting of childhood sickness by socioeconomic status would very likely bias our estimates of the effects of childhood sickness. However, we are identifying the effects of childhood sickness using *within*-family comparisons of brothers. Whatever systematic factors lead people to underreport sickness presumably applied equally to both brothers.

If the effects of childhood health on later life outcomes were the same across families from different socioeconomic groups, then our estimated within-family effect would be unbiased and generalizable to the entire population (again, of white brothers living in families in which one brother was sick and one was not). What if the effects of childhood health varied across socioeconomic groups? We speculate that these effects were weakest among more advantaged families: They had better access to resources that would have allowed their children to recover more quickly from sickness and to avoid its long-term consequences. Consequently, to the extent that there is differential underreporting of childhood sickness across socioeconomic groups, it likely biases our estimates downward (because our sample under-represents people for whom the effect may be largest).

Text strings describing each person's sickness or disability were coded by IPUMS staff using a classification scheme that consisted of several dozen categories. Despite the detailed nature of the coding scheme, about 25% of the sick brothers in our sample had one of just 10 specific maladies, listed here in declining order of frequency: “Measles;” “Injury to Leg, Ankle;” “Fevers (not elsewhere classified);” “Rheumatism;” “Injury to Arm, Hand;” “Malaria;” “Pulmonary Tuberculosis;” “Other Respiratory;” “Paralysis;” and “Back or Spine Disease.” Unlike modern systems for classifying diseases and disabilities, these detailed codes unfortunately represent an admixture of *symptoms* of disease (e.g., “Fevers (not elsewhere classified”)), *causes* of disease (e.g., “Pulmonary Tuberculosis”), and descriptions of limitations that precisely describe neither symptoms nor their causes (e.g., “Back or Spine Disease”).

With all of these conceptual and measurement issues in mind, we have grouped detailed codes into one of four categories, which are listed below (along with the three detailed codes that were most frequently observed within them):

1. *Infectious Diseases:* “Measles,” “Fevers (not elsewhere classified)”, and “Malaria”
2. *Chronic Diseases:* “Rheumatism,” “Paralysis,” and “Back or Spine Disease”
3. *Traumatic Injuries:* “Injury to Leg/Ankle,” “Injury to Arm/Hand,” and “Maimed”
4. *Other Sickness:* “Other Respiratory Diseases,” “Miscellaneous,” and “Diarrhea/ Enteritis”

Because our focus is on within-sibling pair comparisons of socioeconomic outcomes, the only variables that could confound our estimates of the causal impact of childhood health on these dependent variables are those for which siblings differ and may causally affect socioeconomic outcomes. Given literatures on the impact of birth order and sibling spacing on such outcomes (Faurie, Russell, & Lummaa, 2009; Haan, 2010; Lampi & Nordblom, 2010), below we also consider models that adjust for these two characteristics using a dummy variable indicating whether the sick brother is older and a measure of the age difference between the brothers.

Dependent variables

Our dependent variables—literacy, labor force status, occupation and occupational socioeconomic standing, and marital status—are derived from 1900 through 1930 U.S. Census records. That is, we ascertained social and economic outcomes among our 2500 pairs of brothers by linking their 1880 U.S. Census records with their U.S. Census records 20—50 years later; we describe our linking procedures in the appendix. Although we might prefer to also have dependent variables representing other social and economic outcomes (e.g., wealth), the measures we model reflect important aspects of people's economic and social conditions; conclusions based on analyses of these measures speak to theoretical ideas about “social stunting” as described above, albeit in this particular historical content.

The 1900 through 1930 U.S. Censuses obtained consistent and comparable information about each of our specific dependent variables. *Literacy* is a measure of whether men were able to both read and write; we classified men as “not literate” if they were unable to read and/or write. This variable differs from modern measures of literacy in that it does not necessarily reflect *how well* men could read and write. It simply indicates whether men self-reported that they could read and write (presumably *at all*).

For theoretical reasons described above, we are interested in both whether men worked and what sort of work they did. *Labor force status* is simply an indicator of whether any occupation was listed. We classified men as “out of the labor force” if their occupation entry was blank or if it contained an entry that did not describe a line of work (e.g., “none,” “disabled,” “cannot work”). Text strings describing *occupation* were coded to the standards of the 1950 U.S. Census Occupational Classification. Because the decades after 1880 saw the proportion of American men working on farms decline by more than half (from about 50% to about 25%), we began by classifying men according to whether or not they were farming when observed after 1880. We then linked men's detailed occupation codes to four continuous measures of *occupational socioeconomic standing:* (1) occupational prestige (the relative prestige accorded to occupations); (2) occupational education (the level of education typical of occupation incumbents); (3) occupational earnings (the level of earnings typical of

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occupational incumbents); and (4) occupational “status” as expressed using Duncan's (1961) socioeconomic index (SEI, which is a weighted average of occupational education and occupational earnings). See Hauser and Warren (1997) for further descriptions of these measures, each of which has been used in social science and public health research to express the relative social and economic standing of occupations. Finally, we classified each man according to his *marital status* when he was observed at some point between 1900 and 1930.

Results

We present our empirical results in Table 1. In the first row, we report the percentage of men we were able to link across censuses, separately for sick and healthy brothers and separately for all brother pairs (Column A) and for brother pairs in which the sick brother had an infectious disease (Column B), a chronic disease (Column C), a traumatic injury (Column D), or another disease (Column E). Here and below, when we compare sick and healthy brothers with respect to a dichotomous outcome, our statistical inferences are based on conditional logistic regression models that isolate the within-family comparison of interest; because of the logic of this estimator, identification is based only on brother pairs that differ with respect to the dichotomous outcome.

As shown in the first row of Table 1, we were more likely to locate and link records for healthy brothers, regardless of the type of health problem experienced by the sick brother; these differences are statistically significant except for brother pairs in which the sick brother experience a traumatic injury. Based on life table estimates for white males in the late 19th century (described in the online Appendix), we suspect that a large share of the unlinked boys died prior to 1900. In general, our ability to locate healthy brothers did not vary as a function of the disease suffered by his sick counterpart.

In the second row of Table 1, we compare sick and healthy brothers' ability to read and write. Clearly, we can only make this comparison for the 962 pairs in which both brothers were successfully linked; among these pairs, information about one or both brothers' ability to read and write was missing in 19 cases. As shown in Column A of in Table 1, healthy brothers were more likely to be able to read and write than their sick siblings. Although very high percentages of all men in our sample could read and write, sick brothers were 80% more likely than their counterparts to *not* be able to read and write. However, this finding appears to be driven by pairs in which the sick brother suffered from a chronic disease (see Column C).

In the third row of Table 1, we compare sick and healthy brothers with respect to whether any occupation was listed for them on the 1900 through 1930 Census forms; this comparison can also only be made when both members of the brother pair were located. In some instances, no occupation was listed for men or the occupation entry included a non-work activity like “crippled” or “too sick to work” or “doesn't work.” In Table 1 we show that sick boys were less likely to have an occupation listed 20–50 years later. Like the ability to read and write, this result is driven by the pairs of brothers in which the sick brother had a chronic disease, as shown in Column C.

We next compare brothers with respect to the kind of work that they were doing; this comparison can be made for the 735 pairs in which both brothers were located and had some occupation listed. Given the large-scale transformation of the U.S. economy—off of farms and into urban manufacturing jobs—that was underway in 1880, we begin by comparing brothers with respect to whether they were farmers when located between 1900 and 1930. Although the point estimates suggest that sick brothers were modestly more likely to have been farmers, these differences are never statistically significant. Next, we compare brothers in terms of occupational socioeconomic standing (i.e., occupational prestige, occupation education, occupation earnings, and occupational SEI). For these continuous measures of occupational standing, our within-pair comparisons are made using paired *t*-tests with family fixed effects. Here, we find no discernable pattern of results, and differences between sick and healthy brothers are never statistically significant.

In the final row of Table 1, we compare sick and healthy brothers with respect to whether they ever married—that is, whether they were currently married or previously married (i.e., divorced or widowed) when they were observed between 1900 and 1930. Among all pairs, sick brothers were less likely to have ever married; this result appears to be driven by sick brothers with chronic diseases and other illnesses, as shown in Columns C and E.

In general, sick brothers—and especially sick brothers with chronic diseases—were less likely to be linked across censuses (and thus probably more likely to be dead by 1900). Within pairs in which both brothers could be located and linked, sick brothers—especially chronically sick brothers—were less likely to be literate, less likely to have ever married, and less likely to have any occupation reported. However, conditional on having an occupation, sick and healthy brothers engaged in similar kinds of work. Although we are unable to assess formally the impact of selective mortality on our estimates, it seems safe to assume that sick brothers' higher rates of mortality downwardly biases our within-family estimates of the impact of childhood sickness on these outcomes.

Robustness checks

There are at least three potential threats to the internal validity of the findings reported above. First, because extended family living arrangements were more common in the late 19th century and because we do not restrict the ages of the brothers in our sample, it may be that what we are calling sick children were actually sick young adults. For those young adults, sickness may be the result (and not the cause) of social and economic misfortunes. To investigate this possibility, we re-estimated our models after excluding brother pairs in which either brother was beyond age 16. These results appear in Column B of Table 2, and look very much like those for the full sample (albeit with less statistical power).

Second, because socioeconomic outcomes for brothers may have been observed in different census years, there is some risk that what we are calling effects of childhood health may actually be effects of age. However, we have three reasons for not believing that this significantly affects our results. First, about half the time, we observed sick and healthy brothers in the same census (and about 75% of the time, the brothers were observed zero or ten years apart). Second, we observed no general tendency to observe the sick brother earlier or later than his healthy pair. Third, when we re-estimated the models limiting the sample just to pairs in which brothers were located in the same or adjacent censuses, our general findings persist. For example, Column C of Table 2 includes results for analyses in which both brothers in each pair were located in 1920 or 1930.

Third, if sick brothers are systematically older or younger than their healthy siblings are, then the effects of childhood health may be confounded with age. To investigate this possibility, we have re-estimated our models including controls for birth order and for the difference in ages between the brothers. These additional cova-riates do not change our results; consequently, we focus on result from the more parsimonious specification in Table 1. However, In Columns D and E of Table 2, we have re-estimated our models twice: Once restricting the sample to pairs in which the sick brother is older than the healthy brother and once restricting the sample to pairs in which the sick brother is the same age or younger.

This effectively “controls” for birth order. In general, these results are similar to those for the full sample.

Discussion

Socioeconomic position matters for morbidity, but there is growing evidence that the reverse is also true (Haas, 2006; Palloni et al., 2009). In this article, we contribute to research on health selection in general and “social stunting” in particular by estimating the impact of childhood health problems on adult social and economic outcomes using unique historical data on the United States from the turn of the 19th century. Given that these data were collected in an era prior to both the demographic and epidemiologic transitions in the United States (Omran, 1971), our findings provide some insights—however limited and qualified—into the ways in which childhood health problems may impact socioeconomic outcomes among people currently living in developing countries. Similar to other research that has modeled data on siblings (in modern developed countries) to fully account for unobserved factors that might confound relationships between childhood health and subsequent outcomes, we find that childhood health matters for a variety of important outcomes. Boys who were sick in 1880 were probably more likely to have died by 1900. Compared to their brothers, “sick” boys were less likely to be literate, to have an occupation, or to have ever married. These findings are consistent with the hypotheses we derived from the “social stunting” theoretical perspective.

As compared to data collected in the United States and other developed countries in the late 20th and early 21st centuries, our data uniquely contain large numbers of boys suffering from infectious diseases and traumatic disabilities that are now rare. Our research design—based on within-family comparisons—does not allow for formal tests of the relative effect sizes of different types of diseases and disabilities; these would require a set of formal between-family comparisons. For example, we cannot formally compare the magnitudes of the effects of infectious and chronic diseases. Even so, our results provide interesting informal evidence in this regard. First, as noted above, our results about the consequences of childhood illness seem to be driven by boys with chronic illnesses. Second, as shown in Table 1, healthy brothers' outcomes do not appear to vary much across types of maladies suffered by their sick brethren. If infectious diseases have large effects on children's subsequent social and economic attainments, then we would expect to observe that healthy brothers of boys with infectious diseases do worse than healthy boys whose brothers had other types of diseases. This is because those healthy brothers may have contracted those infectious diseases themselves (either before or after the 1880 enumeration). All of this implies that the impact of infectious diseases is either smaller or shorter-lived, whereas the effects of chronic diseases are more pronounced or longer lasting. These findings are also in line with the “social stunting” perspective, which anticipates that early life health problems negatively affect adult social and economic outcomes. In future work we plan to more fully investigate the relative effect sizes of different disease types (using a between family design).

Although our data are unique in a number of positive ways, they also limit the scope of our findings in others. We can generalize only to the population of white men because we did not have the means to link records for women or for racial/ethnic minorities. Because we only observe sickness at one point in time for an age heterogeneous sample of youth, we can say nothing about the ways in which the effects of childhood sickness vary as a function of the stage of the adolescent life course in which it occurs. Although our rates of record linkage are at least as high as in previous research, they are not perfect and this probably introduces some loss of external validity. Selective mortality may bias our estimates and is thus a limitation. However, given that we still find significant effects it is likely that our results actually underestimate the true effects. If boys who would have had relatively worse outcomes were more likely to die before those outcomes could be observed (which seems likely), then our estimates are biased downwards. This may be especially true for the observed effects of chronic illnesses given our relative lack of success in finding sick brothers with chronic diseases. In general, we are confident in concluding that childhood sicknesses—perhaps especially chronic diseases—affect white men's literacy, labor force involvement, and chances of marrying. We recognize the value in replicating our research among women and racial/ethnic minorities, and in such a way that would allow us to consider in more nuanced fashion the ways in which sickness, human capital accumulation, and socioeconomic achievements play out over the life course. However, current data limitations may preclude such investigations.

Nonetheless, our work suggests that childhood health matters for adult social and economic attainments in the context of countries that have not yet fully experienced the demographic or epidemiological transition and are still in the process of industrializing. These findings build on other work on the long-term impacts of malnutrition in the contemporary developing world (e.g., Field et al., 2009; Maluccio et al., 2009). Specifically, we distinguish the effect that *types* of childhood illnesses or accidents had on later socioeconomic outcomes; in so doing, we identify the importance of chronic illnesses during childhood. In spite of the significant public health importance of ameliorating the impact of infectious illnesses in developing countries, our findings highlight the need to also consider the long-term impacts of chronic illnesses on adult status attainment and social mobility.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Socioeconomic outcomes for healthy and sick brothers, by sickness type.

	(A)			(B)			(C)			(D)			(E)		
	All brother pairs			**Pairs in which sick brother has infectious disease**			**Pairs in which sick brother has chronic disease**			**Pairs in which sick brother has traumatic injury**			**Pairs in which sick brother has other sickness**		
	Brother is…			**Brother is…**			**Brother is…**			**Brother is…**			**Brother is…**		
	…Healthy	**…Sick**		**…Healthy**	**…Sick**		**…Healthy**	**…Sick**		**Healthy**	**…Sick**		**…Healthy**	**…Sick**	
Brother's record linked	**66%**	**56%**	**$p < 0.01$**	**66%**	**61%**	**$P = 0.03$**	**64%**	**49%**	**$P < 0.01$**	69%	64%	$P = 0.08$	**68%**	**54%**	**$P < 0.01$**
	(n = 2148 pairs)			(n = 843 pairs)			(n = 675 pairs)			(n = 319 pairs)			(n = 295 pairs)		
Cannot read and write	**3.0%**	**5.4%**	**$P = 0.01$**	3.1%	3.8%	$P = 0.47$	3.5%	10.8%	**$P < 0.01$**	3.0%	1.2%	$P = 0.22$	1.6%	4.7%	$P = 0.18$
	(n = 943 pairs)			(n = 390 pairs)			(n = 259 pairs)			(n = 165 pairs)			(n = 129 pairs)		
Out of the labor force	**10.1%**	**15.7%**	**$p < 0.01$**	8.1%	9.4%	$p = 0.52$	**13.1%**	**26.1%**	**$p < 0.01$**	8.8%	15.3%	$p = 0.06$	11.6%	14.0%	$p = 0.53$
	(n = 962 pairs)			(n = 395 pairs)			(n = 268 pairs)			(n = 170 pairs)			(n = 129 pairs)		
Occupational standing	(n = 735 pairs)			(n = 330 pairs)			(n = 171 pairs)			(n = 133 pairs)			(n = 101 pairs)		
Farmer	24.8%	27.8%	$p = 0.12$	24.2%	26.4%	$p = 0.44$	26.9%	29.8%	$p = 0.47$	16.5%	24.1%	$p = 0.12$	33.7%	33.7%	$p = 1.00$
Occ. prestige (sd)	37.1 (12.6)	37.2 (12.2)	$p = 0.88$	37.3 (12.6)	37.3 (12.1)	$p = 0.99$	38.4(12.1)	37.9 (12.4)	$p = 0.71$	34.8(12.6)	36.7 (12.4)	$p = 0.18$	37.4(13.1)	36.4(11.9)	$p = 0.55$
Occ. earnings (sd)	44.4 (33.0)	44.3 (32.3)	$p = 0.47$	44.6 (33.2)	41.9 (33.8)	$p = 0.78$	43.7 (33.0)	44.6 (31.2)	$p = 0.38$	42.4 (33.6)	40.0 (31.9)	$p = 0.81$	44.4 (33.0)	43.3 (32.4)	$p = 0.58$
Occ. education (sd)	13.1 (18.4)	12.4 (16.4)	$p = 0.41$	14.1 (19.9)	13.3(17.8)	$p = 0.28$	10.6 (14.6)	12.9(16.2)	$p = 0.70$	13.0 (20.1)	10.6(15.2)	$p = 0.14$	13.1 (18.4)	12.4 (16.4)	$p = 0.22$
Occ SEI (sd)	29.8 (23.9)	29.3 (23.4)	$p = 0.67$	31.2 (23.7)	29.5 (24.0)	$p = 0.89$	27.8 (24.0)	29.5 (23.8)	$p = 0.47$	28.1 (24.2)	26.1 (21.9)	$p = 0.54$	29.8 (23.9)	29.3 (23.4)	$p = 0.50$
Ever married	**83.0%**	**76.1%**	**$p < 0.01$**	83.3%	82.0%	$p = 0.76$	84.3%	**67.2%**	**$p < 0.01$**	75.9%	78.2%	$p = 0.88$	88.4%	73.6%	**$p < 0.01$**
	(n = 962 pairs)			(n= 395 pairs)			(n= 268 pairs)			(n = 170 pairs)			(n = 129 pairs)		

Note: p values are from tests of the null hypothesis that the population mean difference between healthy and sick boys is zero. For dichotomous outcomes (literacy, labor force participation, whether the man is a farmer, and marital status), these tests were performed using family fixed effects logit models. For continuous outcomes (occupational standing), these tests were performed using paired *t*-tests. Bolded values indicate that the null hypothesis is rejected at the 0.05 level.

Table 2

Socioeconomic outcomes for healthy and sick brothers, by sickness type.

	(A)			(B)			(C)			(D)			(E)		
	All brother pairs (Repeated from Column A, Table 1)			**Brother pairs in which both are 16 or younger**			**Brother pairs ; in which both were located in 1920 or 1930**			**Brother pairs in which the sick brother was older**			**Brother pairs in which the sick brother was not older**		
	Brother is…			**Brother is…**			**Brother is…**			**Brother is…**			**Brother is…**		
	…Healthy	**…Sick**		**…Healthy**	**…Sick**		**…Healthy**	**…Sick**		**…Healthy**	**…Sick**		**…Healthy**	**…Sick**	
Cannot read and write	**3.0%**	**5.4%**	***p* = 0.01**	2.3%	4.2%	*p* = 0.06	**2.0%**	**4.4%**	***p* = 0.02**	2.9%	5.0%	*p* = 0.07	**3.1%**	**5.9%**	***p* = 0.03**
	(*n* = 943 pairs)			(*n* = 478 pairs)			(*n* = 453 pairs)			(*n* = 517 pairs)			(*n* = 426 pairs)		
Out of the labor force	**10.1%**	**15.7%**	***p* < 0.01**	6.1°%	9.4%	*p* = 0.06	**9.5%**	**16.2%**	***p* < 0.01**	**8.2%**	**19.4%**	***p*< 0.0l**	12.4%	11.3%	*p* = 0.59
	(*n* = 962 pairs)			(*n* = 490 pairs)			(*n* = 463 pairs)			(*n* = 527 pairs)			(*n* = 435 pairs)		
Occupational standing	(*n* = 735 pairs)			(*n* = 417 pairs)			(*n* = 357 pairs)			(*n* = 357 pairs)			(*n* = 357 pairs)		
Farmer	24.8%	27.8%	*p* = 0.12	**22.1%**	**27.1%**	*p* = **0.05**	25.8%	29.4%	*p* = 0.20	**24.3%**	**31.2%**	***p*= 0.02**	25.2%	23.8%	*p* = 0.58
Occ. prestige (sd)	37.1 (12.6)	37.2 (12.2)	*p* = 0.88	36.9 (12.4)	37.4 (12 .0)	*p* = 0.55	37.7(12.1)	37.9(12.1)	*p* = 0.85	36.7 (12.7)	37.1 (11.5)	*p* = 0.61	37.7 (12.4)	37.4 (12.9)	*p* = 0.47
Occ. earnings (sd)	44.4 (33.0)	44.3 (32.3)	*p* = 0.47	45.3 (32.6)	43.7 (32.5)	*p* = 0.40	46.5 (33.7)	43.1 (33.3)	*p* = 0.12	43.5 (33.1)	41.4 (32.0)	*p* = 0.29	45.3 (32.9)	45.5 (32.7)	*p* = 0.82
Occ. education (sd)	13.1 (18.4)	12.4 (16.4)	*p* = 0.41	13.0 (18.1)	11.9 (16.0)	*p* = 0.25	12.9(17.9)	12.9(17.0)	*p* = 0.99	12.7 (17.4)	11.4 (14.8)	*p* = 0.22	13.6(19.5)	13.6(18.1)	*p* = 0.82
Occ. SEI (sd)	29.8 (23.9)	29.3 (23.4)	*p* = 0.67	29.8 (23.5)	29.2 (22.9)	*p* = 0.64	30.4 (24.3)	30.2 (24.0)	*p* = 0.89	29.4 (23.8)	27.6 (22.1)	*p* = 0.23	30.2(24.1)	31.3 (24.7)	*p* = 0.69
Ever married	**83.0%**	**76.1%**	***p* < 0.01**	81.0%	78.0%	*p* = 0.20	**90.5%**	**86.0%**	***p* = 0.02**	**82.2%**	**77.4%**	***p*= 0.04**	**83.9%**	**74.5%**	***p* < 0.01**
	(*n* = 962 pairs)			(*n* = 490 pairs)			(*n* = 463 pairs)			(*n* = 527 pairs)			(*n* = 435 pairs)		

Note: p values are from tests of the null hypothesis that the population mean difference between healthy and sick boys is zero. For dichotomous outcomes (literacy, labor force participation, whether the man is a farmer, and marital status), these tests were performed using family fixed effects logit models. For continuous outcomes (occupational standing), these tests were performed using paired t-tests. Bolded values indicate that the null hypothesis is rejected at the 0.05 level.